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Re: The Relationship Between Premature Ejaculation and Hyperthyroidism

Cihan A, Demir O, Demir T, Aslan G, Comlekci A, Esen A.

J Urol. 2009;181:1273–80

Expert's summary:

Cihan and coworkers studied the prevalence and characteristics of premature ejaculation (PE) in a single-center prospective study in a small Turkish population of hyperthyroid subjects ($n = 43$; 40% with Graves-Basedow disease, the rest of the sample with toxic nodules). PE was defined according to *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* criteria along with patient-reported outcome and stopwatch measurement of intravaginal ejaculation latency time (IELT; PE: IELT of <1 min). PE was observed in 31 (72%) hyperthyroid subjects. There was a positive correlation between thyroid-stimulating hormone (TSH) and IELT. After 2 mo of high-dose medical therapy, subjects were enrolled in continuing medical therapy ($n = 10$) or had definitive treatment with surgery ($n = 7$) or radioactive iodine ($n = 7$). Achieving euthyroidism at follow-up (24 patients), regardless of therapeutic intervention, increased mean IELT by a factor of 2 in the total population or by a factor of 3 in the PE population. Beck anxiety scores and International Index of Erectile Function also significantly improved in the euthyroid state. The authors conclude that hyperthyroidism should be considered a novel and reversible etiological risk factor for PE.

Expert's comments:

Although both PE and hyperthyroidism are relatively common conditions, their association has not been systematically studied until recently. In a consecutive series of 755 men presenting with sexual dysfunction, a 2-fold greater prevalence of hyperthyroidism was evident among men

with PE [1]. According to this finding, Carani et al [2] demonstrated in a small, multicenter, prospective study that most (50%) hyperthyroid patients have PE. This prevalence was substantially reduced (15%) by treating the underlying disease, with a consequent doubling of ejaculatory latency. In Cihan et al as well as in Corona et al [1], a role for hyperthyroidism-induced anxiety was also suggested as a cause for PE; however, at multivariate analysis, even after adjusting for anxiety, low TSH was independently predictive of PE [1]. Inducing an experimental hyperthyroidism in male rats was associated with enhanced seminal vesicle contraction and activity of bulbospongiosus muscle [3]. These findings suggest a direct role for thyroid hormones in decreasing ejaculation latency that is independent from hyperthyroidism-induced anxiety. Furthermore, Carani et al [2] showed that medical treatment of the opposite state, hypothyroidism, resulted in a 2-fold decrease in ejaculatory latency and a reduction in delayed ejaculation. Hence, the view that thyroid hormones regulate not only the ankle reflex but also the ejaculatory reflex is consistently emerging [4].

Conflicts of interest: The author has nothing to disclose.

References

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- [3] Cihan A, et al. J Urol 2009;181:907–12.
- [4] Donatucci CF. J Sex Med 2006;3(Suppl 4):303–8.

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E-mail address: m.maggi@dfc.unifi.itDOI: [10.1016/j.eururo.2009.08.006](https://doi.org/10.1016/j.eururo.2009.08.006)**Re: Aberrant ERG Expression Cooperates with Loss of PTEN to Promote Cancer Progression in the Prostate**

Carver BS, Tran J, Gopalan A, et al.

Nat Genet 2009;41:619–24

Expert's summary:

The authors investigated the functional consequences of Ets-related gene (ERG) rearrangement and overexpression and its association with phosphatase and tensin homolog (PTEN) loss in prostate cancer (PCa). PTEN was lost in 14 of 15 ERG-rearranged samples but was lost in only 13 of 25 nonrearranged samples, suggesting that PTEN loss and ERG genetic rearrangements are concomitant events in PCa. Mouse experiments showed that combined ERG overexpression and PTEN haploinsufficiency lead to accelerated development of high-grade prostatic intraepithelial neoplasia (HGPIN) and subsequent progression to multifocal, invasive PCa. In contrast, ERG overexpression alone had no effect in

mice with normal PTEN status. Functional experiments revealed that ERG overexpression increases cell migration through upregulation of the two genes chemokine (C-X-C motif) receptor 4 (CXCR4) and ADAM metalloproteinase with thrombospondin type 1 motif, 1 (ADAMTS1), while PTEN haploinsufficiency is known to promote cell proliferation through activation of protein kinase B (Akt). The authors concluded that ERG rearrangements and PTEN loss are frequent concomitant events in PCa, and that this cooperation promotes progression of HGPIN to invasive cancer.

Expert's comments:

This work continues the exciting story of transmembrane protease, serine 2 (TMPRSS2)–ERG rearrangement in PCa [1]. The TMPRSS2–ERG gene fusion prevails in almost 50% of PCa and leads to ERG overexpression through activation of the androgen receptor–regulated gene promoter of TMPRSS2. Despite the high prevalence of TMPRSS2–ERG rearrangement, its biological role has remained unclear. In accordance with